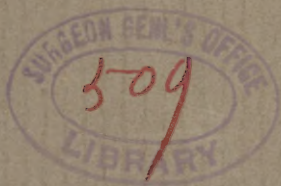


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THE FORMS OF PERITO-
NITIS, THEIR RELATION
TO APPENDICITIS, AND
THE ÆTIOLOGY OF
EACH.

BY

ROSWELL PARK, A.M., M.D.



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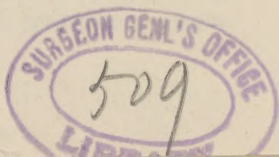
BY ROSWELL PARK, A.M., M.D.†

If I venture upon this occasion to endeavor to interest you in a subject which has already been hackneyed, it is because I hope to be able to present it in a way not commonly adopted by writers on the subject. It is less with the symptoms and treatment of the various forms of local and general peritonitis that I care to deal than with certain features in its ætiology.

While every competent practitioner is expected to make a diagnosis of peritonitis, it has yet happened to me so frequently to find that they are misled, or at least that they have overlooked the primary development of the local focus of infection or inflammation at the site of the appendix, from which the general condition has extended,—and that they have contented themselves with treating the sequel and neglected the primary condition,—that I have been tempted to take up the subject from this aspect.

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In a paper prepared some time ago for the "System of Practical Therapeutics" edited by Dr. Hare (Vol. II, pp. 1015 *et seq.*) I have ventured to give a classification of various forms of peritonitis, which, if not original, is at least more comprehensive than any which up to that time had been found in the ordinary text-books or works of reference. Subsequent experience has served to confirm my impression that the classification therein given is satisfactory save in one or two regards, and it is with an amended presentation of the matter at that time presented that I shall first trouble you to-night.

I have ventured to classify the different forms of peritonitis in three ways: First, clinically; second, anatomically; and third, pathologically.

Let us take first the clinical classification, making the following divisions, according to authorities which have long been considered competent:

1. *Idiopathic Peritonitis*.—If there is anything which modern research has taught us regarding abdominal complaints, it is, I think, this: that there is no such thing as a purely idiopathic peritonitis. This term, therefore, must really be taken to mean an occurrence of the disease for which no known origin can be ascribed; in other words, the term "idiopathic" is a confession of ignorance concerning causes. This so-called

idiopathic peritonitis is known, *e.g.*, in the course of Bright's disease and of scurvy. In the former there is undoubtedly a chronic toxæmia, and there is now very good reason for considering the latter to be a disease of bacterial origin, especially since the most recent observations of Babes. The exact relations of the various toxæmias to serous inflammations are not yet known, but it will be my purpose here to-night to show that toxæmias of all kinds predispose to colon infection, or in fact to any other active infectious process. If there be such a thing as a true rheumatic peritonitis it is due to the unknown poison of rheumatism, be this what it may, and in that case is not idiopathic. Why in the course of acute rheumatism the peritoneum and pleura should suffer so rarely and other serous membranes so often, we do not know. Malaria may certainly cause congestive disturbances in the peritoneum, and these are frequently serious enough to call for active treatment, and may very easily predispose to active colon infection. The congestions and neurotic disturbances due to malaria yield to quinine, but an active, distinct peritonitis due to malaria cannot yield to quinine alone, in my estimation, since such a condition is one of infection.

In times past among the forms of idiopathic peritonitis has been recognized a "menstrual" form in which probable exposure

to cold plays a certain part, which if it go on to exudation and possibly suppuration must be positively due to bacterial infection.

2. *The Consecutive Form.*—This term has been applied where there has been evident inflammatory extension from certain parts, due to inflammation, and most frequently from the abdominal viscera. It appears furthermore that this extension takes place more often from the hollow than from the solid viscera.

For instance, it is much more common as an extension from the stomach, intestines, gall-bladder, uterus, or Fallopian tubes, than from the spleen, liver, kidneys, pancreas, or mesenteric lymph nodes. It is also consecutive to certain parietal inflammations, erysipelas, carbuncles, and burns of the abdominal wall. Less often it succeeds inflammations of the thoracic viscera: for example, pleurisy, pericarditis, pulmonary abscess, empyema, hydatid cyst, and even phrenitis. It may follow infectious embolism of the abdominal aorta; it may be consecutive to phlebitis, or periphlebitis of the abdominal pelvic veins; it comes on after infiltration from any cause. Further, the most common instances of consecutive peritonitis are met with after strangulation of the intestines, or obstruction from any cause, and also from the results of puerperal conditions. So far as the former are concerned, a small strangulated hernia, in-

vagination of the bowels, or fæcal impaction producing obstruction, are usually favoring anatomical conditions. In any one of these instances the process consists first of stasis, then exudation, and then infection. Of the condition formerly known as puerperal peritonitis, it must be said that it is even more common after abortion or miscarriage than after delivery at term.

3. *Perforative Form.*—That peritonitis frequently ensues after the escape into the peritoneal cavity of any secretory or excretory material which Nature has intended to keep out of it, is well known. To this general rule there are, however, such exceptions as those for instance, when bile or pancreatic fluid escape into the general peritoneal cavity, and occasionally urine also. Such cases are for the most part fatal, although in rare cases the inflammation is circumscribed and recovery may ensue. Perforation may occur from the stomach, as in cases of gastric ulcer, and these are the cases where a localization of the inflammation is most probable; it most often occurs from the intestine. The majority of these cases are post-typhoidal. The disease assumes a fulminating type, and patients die very rapidly. It may be the result of post-dysenteric lesions, when there has been a membranous form of enteritis, in which there is formation of pus and sloughing of a portion of the intestinal wall. It may

be the result of gangrene due to strangulation of the gut, with sloughing and a faecal extravasation. When a hydatid cyst ruptures into the peritoneal cavity it nearly always causes acute peritonitis, its contents being absolutely toxic. Perforative peritonitis may also be the result of rupture of an ureter, this being usually due to the presence of a calculus; or to rupture of the bladder or gall-bladder, or of any abscess, for example pericæcal, in the neighborhood of this membrane. The onset of cases due to perforation is acute, and is followed usually by a sudden meteorism with profound collapse and speedy death.

4. *Traumatic Form*.—This combines all cases due primarily to trauma, whether the peritoneum has been penetrated or not. Violent, continuous pain in the abdomen is not infrequently provoked by it, while crushing injuries which produce rupture of the liver or of the kidneys are not infrequently followed by it. The most common injuries which produce it are, however, penetrating wounds, lacerated, punctured and gunshot.

5. *Chronic Peritonitis*.—This form is perhaps more often due to a condition of chronic Bright's disease than to any other one factor. It has but little in common with the acute form, perhaps nothing save identity of location. Ordinarily it is not an infectious condition nor the result of it. It is due rather

to the constant irritation of some toxic agent. Anatomically it is characterized by a much thickened and opaque peritoneum within which, more and more, adhesions have formed. The amount and extent of these adhesions in some cases are wonderful. Cases are on record in which all the viscera were so matted together as to be simply inseparable. (*Peritonitis obliterans seu deformans*.) This clinical form is characterized by the absence of acute manifestations and a tendency to fibrinous exudate which in time often forms adventitious membranes that later constitute separate cavities, all of which causes the peritoneum to appear larger and thicker than it really is. This appears to be noted most often about the liver, and it is worth while to remember that in cases of so-called peri-hepatitis the thickened condition of the liver is in a large measure due to this cause.

These new membranes may divide the general peritoneal cavity into a few or numerous chambers so that when the abdomen is opened it appears to be filled with cysts; inasmuch as fluid collects in each of these chambers the resemblance is more striking. It is characteristic of chronic peritonitis that the mesentery is not only shortened, but that the actual length, by measurement, of the intestine is diminished sometimes even by several feet, while its diameter is correspond-

ingly contracted. This is the result of cicatricial or atrophic changes.

This form is nearly always accompanied by marasmus, which may be explained by the anatomical condition just alluded to and by more or less ascitic collection of fluid. It sometimes is very permanent. On the other hand the peritoneal condition itself is sometimes the result of ascites due to other causes, since the distension of the membrane by fluid seems to lead to chronic inflammatory processes in its texture. The former condition corresponds to the idiopathic dropsy of many of the older writers. From what has been said it appears that this form is not necessarily a chronic process, but may be the sequel of an acute attack.

6. *Tubercular Peritonitis*.—Like other tubercular lesions, this may be primary or secondary. The slow forms are distinguished by the condition of the peritoneum just described, *plus* the peculiar dotted or nodular appearance which tubercular deposits produce. In many of these cases, especially the slow ones, the lymph nodes will be found to have undergone caseous alteration. The abdominal cavity may contain serum, clear or turbid, pus or puruloid material, or recent or old blood, since hæmorrhage more frequently results from surface lesions. Tubercular peritonitis assumes sometimes a proliferative type and is characterized by

numerous adhesions. It may be acute, in fact even as acute as tubercular meningitis, or it may follow a very slow and irregular course. Its symptoms are vague and obscure; its characteristics are those of general ill health, which later becomes pronounced marasmus accompanied by diarrhœa, especially if the intestines be ulcerated. Unless pus and fluid be present the abdominal walls are retracted. The signs of this condition are induration and nodulation of the omentum when it can be felt, *and reddening and thickening around the umbilicus*, which are due to extension along the obliterated umbilical vessels.

7. *Malignant Peritonitis*.—Less than one-half of these cases are really primary; the majority appear to be secondary. There is known a primary miliary carcinoma, but it is very rare; as a secondary condition it is less so. Circumscribed cancer of the peritoneum is known, and is met with occasionally. The abdominal cavity is filled with a mass of colloid material to which the name colloid cancer has been given, although it is really due to colloid degeneration of medullary carcinoma, or small round-cell sarcoma which is most likely to occur in the omentum. This form is characterized by mildness of symptoms, by a progressively downward course, sometimes by hæmorrhage which may prove fatal. Recent investigations make

it probable that in every case of widespread cancerous disease a peculiar toxæmic condition is met with which is due to an unknown toxine produced by abnormal tissue changes.

8. *So-called Latent Form of Peritonitis.*— Every practitioner of wide experience meets with fatal cases in which pus or other evidences of diffuse or local peritonitis are found, but in which during life such a condition of affairs had not been suspected. Such instances are most common in connection with appendicitis. This so-called latent form is nearly always characterized by the presence of pus, and is simply an evidence of failure in diagnosis, such failure being sometimes due to profundity of the toxic symptoms, sometimes to diversion of attention by other lesions, and sometimes to the fact that when pus forms very rapidly the exquisite sensibility of the peritoneum is lost. This latent form of disease deserves no place in pathology, and should be clinically classed only as a curiosity.

9. *The So-called Fœtal and Uterine Form.*— That congenital stenosis of the intestine may lead to fatal peritonitis has been shown by Virchow and others. It is usually readily fatal from the nature of the condition which primarily produced it. An inflammatory form has also been distinguished as occurring shortly after birth, and is for the most part really a form of consecutive peritonitis.

being due to causes arising about the umbilicus, such as inflammation, lymphangitis, gangrene of the umbilical vessel, or umbilical hernia. It is for the most part of septic type, possibly even putrid.

I have given a clinical classification first, because hitherto it has been so generally regarded as important. It is based on external appearances, and these are notoriously often misleading. It is not a scientific classification, although I confess that it has answered a certain purpose in time past.

The anatomical classification is very simple. It is as follows:

1. *The General or Diffuse Form*, which has already been sufficiently considered.

2. *The Circumscribed or Localized Form*, which may merge into the first as congestion is excited, and exudate transplanted by means of the respiratory and peristaltic motions to which the abdominal contents are subjected. The most common examples of the circumscribed form are met with upon the bowels and about the cœcum. The traumatic form clinically is frequently represented by the circumscribed form anatomically, especially when of low grade. A very rare form of the disease is that which is limited to the cavity of the lesser omentum, and which is for the most part consecutive to disease of the pancreas. Virchow has also described a *peri-*

tonitis mesenterialis et omentalis. Localized peritoneal exudate is also frequently due to faecal accumulation and to the presence of cystic tumors which become infected and give rise to local as well as general processes of an infectious type.

Before discussing the pathological classification let me stop a moment and make a little clearer my position. It is my object to demonstrate that in reality there is no such thing as an idiopathic peritonitis. In order to do that, it is necessary first to maintain that peritonitis properly speaking is an infectious disease—in other words, a disease or a condition having a bacterial origin; in the second place, that for every case of this nature a path or at least a focus of infection must be made out; and third, that we are now in position to claim that *for all such cases when properly studied such path or source of infection can be made out*.

Were I addressing a less intelligent audience I might think it necessary to go perhaps further back, and show that all supuration has its source in actual germ activity, and that all putrefactions are conditions brought about by bacterial action, and that consequently there can be no such thing as pus or suppuration, nor ichor, nor any putrid condition of affairs in the abdominal cavity, save those which are produced di-

rectly by some bacterium. Injection of merely irritating material into the peritoneum, so long as infection be prevented, may produce congestion, exudate, hæmorrhage, or even death of tissue, and yet not cause true peritonitis. Or, as is more often the case, such materials pave the way for subsequent infections, usually from the intestinal canal, and thus bring about a distinct peritonitis. But it is a part of my purpose here to maintain that mere congestion or innocent exudate does *not* bring about either the clinical signs of peritonitis or the pathological condition which is a part of the genuine disease. In fact, so completely protected are we, under circumstances of healthy surroundings by the natural properties of the peritoneum, that it has been shown by many investigations that even a direct or indirect introduction into the peritoneal cavity of pyogenic organisms can set up an acute peritonitis only providing there be present some local predisposition.

Grawitz, for example, has shown that in dogs, if pyogenic organisms along with an absorbable amount of fluid be injected into the normal peritoneal cavity, peritonitis is produced only when they are introduced in excessive amount, or when simultaneously there occurs or is present some necrosis of tissue by which the pathway is prepared for their penetration into the deeper layers of

the serosa, or when some wound of the abdominal wall favors localization of infection. (Charité Annalen, 1886.) In another place I have more thoroughly discussed the pathological side of this question, and prefer not to weary you with all of it here. (See my Mütter Lectures on Surgical Pathology, page 135 *et seq.*)

Let me add only that Cheyne has shown that suppurative peritonitis occurs with the greatest certainty when there is a wound or some other focus, in or near the abdominal wall, from which infection can occur, and from which as a centre organisms are constantly being passed into the cavities themselves. This is still more certain to occur if the wound be an unhealthy one. For example, in rupture of the healthy bowels if the extravasated contents are early thoroughly removed, and the edges of wound approximated, recovery commonly occurs; but if the perforation of the bowel wall takes place in an unhealthy area, there has been already established a nidus in which organisms have been growing, and presumably in their more virulent forms.

With these remarks we are now prepared to take up the pathological classification, which I have modeled after that of Bumm.

1. *Aseptic Form*, usually local, not always. Characterized by hæmorrhagic and fibrinous exudate with strong adhesive tendency.

2. *Infectious Form*, divided into (a) *staphylococcus* and *streptococcus forms*—the latter are usually post-puerperal or are connected with pelvic diseases of women, and are more rapidly fatal; (b) *colon infection*, due to the *bacillus coli communis*; (c) *septic*, characterized by pus instead of ichor, and by acute onset with chill and pyrexia; (d) *putrid*, usually the result of the perforative forms.

3. *Specific Forms*, tubercular, actinomycotic, and gonorrhœal, if there really be such a specific form.

(Flexner has recently described a case of peritonitis where the infection was due to the *proteus vulgaris*. So far as I know, this is the only instance of its kind and hardly deserves to be classified as yet in one or the other of the above forms. For all we know, there may be other bacteria which have at one time or another a power of setting up the same condition. Such cases, however, are necessarily so rare that in such a discussion as this we may omit all further reference to them. Suffice it that for our present purpose the second, or infectious form, is that with which we particularly have to deal at present.)

Going into a little more detail, it may be said of the first, the *aseptic* form, that it is at least rare, though experiment has shown that it is possible to provoke congestion, or exudation, or even hæmorrhage into the peritoneal

cavity, which shall be accompanied by certain symptoms and shall not assume an infectious type. These cases are sometimes post-operative, but most often they are produced by contusions or external injuries.

The *infectious* forms are those which most interest us here. It has been shown that pyogenic organisms produce peritonitis for the most part when introduced in excessive amount, or when some other substance is present and assists their penetration into the deeper tissue layers, or especially when some previous lesions favor the localization of the infection. The first, the *streptococcus* and *staphylococcus* cases, are usually met with after parturition or abortion. They occur also after acute or recurring tubal or ovarian disease, and on section there is found a thin puruloid, odorless exudate, or if late this may be thick and creamy. Early in the disease this exudate is extremely infectious; it loses its virulence as it progresses or in proportion to its slowness.

We have spoken above of a gonorrhœal form of peritonitis as belonging to the specific type. This is in deference to certain authorities whose statements we nevertheless feel inclined to challenge. So far as the writer has been able to study the question, a pure type of distinct gonorrhœal infection, by natural channels, is almost an impossibility. Most of these cases are really

instances of mixed infection, the gonococci being almost invariably associated with staphylococci or streptococci. It surely is so in the urethra, and is probably so in pelvic infections in women which are ascribed to previous or latent gonorrhœa. With these cases, in this place, we have nothing further to do.

Peritonitis due to infection from the *colon* is a variety recently established, mainly by the investigations of Welch and his colleagues in the Johns Hopkins Hospital. By their investigations and those of others it has been completely demonstrated that under certain conditions, not yet completely understood, the colon bacillus escapes from its ordinary habitat, and is capable of producing intense and even fatal disturbance, not only in the peritoneum, but in widely distant parts of the system. For a moment I wish to drop its further consideration and discuss the other infectious forms of peritonitis, namely, the *septic* and the *putrid*. The former is probably also an instance of colon infection mixed with other forms. As already stated, it is characterized by pus instead of ichor, and by a sharp onset with chill and pyrexia. The putrid form is that which occurs most commonly after operations for perforation. It begins with a chill, but with fever which gradually augments, and is characterized by a putrid, foul-smelling exudate. It is more

infectious than the other forms and contains a mixture of micro-organisms, many of which are known to be non-pathogenic, and some of which at least are saprophytic; in other words, it is the result in large measure of putrefactive organisms, while the pyrexia which accompanies the condition is due chiefly to the ptomaines which they produce, and the decomposition of the exudate is due to their well known destructive properties.

Recurring now to the colon bacillus, with which this paper largely deals, let me go over briefly some of its most salient features. It is known to be an inhabitant of the alimentary canal, where it probably has more or less to do with the breaking up of food products, *i.e.*, digestion. In the healthy intestinal canal it never exists alone, but is probably always mingled with other organisms, with some of which it may enjoy a sort of commensalism. It has been abundantly shown by various observers that this organism has the power of passing through the uninjured intestinal wall and peritoneum. The fact is established, although the mechanism of its passage is not known. One of the most significant features in its life history is that within wide limits it varies in virulence and that in its ordinary state it is usually not virulent. This will account for the slightly infectious property of the pus met with in certain intra-abdominal abscesses. This will

account, too, for the fact stated by many surgical observers, that pus from the gall-bladder, and pus from many pericæcal abscesses, when accidentally allowed to escape into the peritoneal cavity, seems often to be devoid of poisonous properties. That it varies in virulence within extensive limits is most certain. This variation finds its highest pathogenic expression in cases of cholera nostras, since when recovered from these and inoculated it usually causes death from acute septic infection within twenty-four hours. This virulence is best retained in bouillon cultures; those on agar lose it much more speedily, and virulent cultures may thus be attenuated. When the colon bacillus manifests pyogenic properties and produces pus, it appears to be because it has become in some measure attenuated. When death occurs and seems due to supuration produced by it, although probably in reality due to an intoxication, the bacilli are rarely found anywhere else save in the pus. So we have to deal with the organism under two conditions—at one time as an active agent producing acute septic infection, at another as a common pyogenic organism producing local abscess.

It is of interest here to state that the colon bacillus is identical with the organism which Passet called the *bacillus pyogenes fætidus*, as well as with the *bacillus lactis aerogenes*,

and with Clado's urinary pyogenic bacterium. In fact, the colon bacillus is the organism in so-called urinary infection. We are not yet in position to state definitely the exact condition under which this organism forsakes the alimentary canal and gains entrance into the kidneys, urethra, or bladder, or passes up the biliary duct into the gall-bladder and into the liver, setting up, as it is known to do, abscesses in both these locations; nor how it may in rare instances produce pleurisy or endocarditis, or even abscess of the brain as I personally have known it to do. But although we cannot explain its migration nor account for its presence, we can at least recognize the latter, and we now know that the colon bacillus is probably the greatest enemy with which the laparotomist has to contend, and we know that without extreme care it is very easy for him to get a post-operative peritonitis which is due to infection of the peritoneum by this bacterium, which is not necessarily fatal, but which is most undesirable and serious, to say the least.

Let us now leave the peritoneum for a little while and consider the appendix. Concerning its anatomical location we are at present not concerned, but it seems to me of very striking importance that we recognize one or two points in its histological construction. It was Bland Sutton who first pointed out to us that on the margin of every such

diverticulum or normal sac there is a collar of lymphoid tissue. We see it, for instance, at the upper end of the œsophagus, in the shape of the faucial and pharyngeal tonsils. It is found moreover in many other places. There is also more or less of this same lymphoid tissue about the opening into, and the tissue of, the appendix. It is inflammable in a high degree, and it is also easily infected. Its character is such, moreover, that when infected, infection may travel easily, and may be both destructive and hard to subdue. By means of the communication which this lymphoid tissue enjoys with the lymph vascular system, it is easy for the infection to migrate—in other words, for it to spread. The appendix is, moreover, so constructed that when once infected, mycotic necrosis is easy. (This is of course accepting Eppinger's views concerning *necrosis epithelialis mykotica*.) That is, from pressure and irritation by fæcal masses, and resulting exudate, there follows easy invasion of sub-epithelial tissue, with extension frequently to the peritoneum. Along with this also goes such stricture of the proximal end and dilatation of the distal end, with retention and vascular stasis, that gangrene often occurs. Another point of no small interest about the appendix is that it is a vestigial relic of a formerly useful organ which in the evolution of the human race is being gradually gotten rid of. That is, it is

now undergoing retrograde changes, and like all such retrogressive tissues or parts it is extremely liable to inflammation, and when inflamed seems to have very little power of vital resistance.

What now are the common clinical causes which lead to appendicitis? For the most part they are connected with atony of the large intestines, with faecal retention, improper function of the mucous membrane, defective secretion, irregular contraction of the muscular coat, and mild forms of chronic colitis. Simon has recently called attention to attenuated forms of this kind of trouble as frequent in young people and especially in children. Dietetic vices are the most common causes of the condition above alluded to. These vices may consist of excessive eating, or eating of improper food, and the gradual establishment of unhygienic habits concerning the emptying of the large bowel. Previous diseases, like typhoid or dysentery, frequently leave behind them certain lesions in the colon which predispose to distension of the intestine and chronic inflammation. They leave sometimes also cicatricial reminders of their presence in the shape of adhesions, contractions, and probably rigidity of the ileo-cæcal valve, all of which are important predisposing causes. (Gambetta's case furnished a remarkable example of this kind.) In other words, these causes, contributing

more or less to the irritative contractility of the intestines and the accumulation of fæcal débris, predispose to appendicitis. So much for the general causes. The more immediate causes connected with this inflammation seems to be inseparable from encroachment of various perivascular exudative infections through the submucosa, and occurrence of ulcers through circulatory disturbance, which may go on to perforation. Or the exudation may be so great as to produce gangrene very early from mere strangulation. It will be remembered that the appendix itself is not supplied with abundant blood-vessels, but has only a single nutrient artery, consequently stasis due to sepsis may easily and early shut off all blood supply. Most of these causes probably begin as endo-appendicitis, after which the necrotic changes quickly follow in the mucosa itself. When we have rapid formation of phlegmon around the inflamed portion, there may be several distinct foci of suppuration, although it is not necessarily always the case. Peritoneal infection probably spreads along that portion of the peritoneum which invests the appendix. We have not yet learned the condition, which in one case permits the formation of adhesions and the limitation of the exudate, or in another permits of widespread infection. That the fibrinous exudate may be very large in amount, the rapid formation of tumor and

sometimes its almost equally rapid disappearance amply proves.

As a matter of fact, however, there are very few of these cases which are not really caused by colon infection, or infection by the colon bacillus. (The researches of Dr. Bristow reported recently by Dr. Fowler in the *Annals of Surgery*, January, 1894, p. 28, show that pure cultures of colon bacilli can be obtained from the peritoneal surface of an inflamed appendix which has not undergone perforation, as well as from non-encysted intra-peritoneal collections of fluid. A chronic condition such as above described, or a more acute one of the same or more outspoken inflammatory character, leads to stasis and such anatomical disturbances in the structures of the appendix itself as to cause them to lose their resistance and to fall a sudden prey to the colon bacillus, whose virulence perhaps has recently been augmented by some disturbance farther up the alimentary canal. There are now countless cases on record to show that we have to do in a large proportion of instances with a primary and perhaps pure infection of this kind. At other times it is undoubtedly mixed, the colon bacillus being mixed with the ordinary pyogenic staphylococci and streptococci. My own impression, although I would prefer not to state it too strongly at present, is that the most virulent or most

rapidly infected cases are those in which we have mixed infection. Still the colon bacillus as we often meet with it is enough to kill many persons, not only in spontaneous cases like those of appendicitis, but in post-operative cases of peritonitis, where not infrequently the pus is found to be a pure culture of this organism.

I want to insist, therefore, upon maintenance of this position—that appendicitis is always due to local infection, which, as in all other parts of the body, may proceed to gangrene. It sometimes happens that this gangrene is so much more rapid than could probably occur by mere cutting off of blood supply that we are compelled to suppose that the infective agents themselves possess a most destructive virulence amounting practically to malignancy.

What now is the relation of appendicitis to peritonitis?

When we consider the anatomical relation of the two structures, it will be seen how easy it is for infection to proceed from one to the other without reference to whether perforation has taken place or not. In other words, peritonitis may follow the non-perforative as well as the perforative form of appendicitis. It depends in some measure upon whether the latter be of pure or mixed type. At times the virulent products which are poured out in the peritoneum are so ichorous

and so profuse that patients die in collapse without excessive meteorism, or without those general appearances which would give the average practitioner to understand what has occurred. At other times the infection of the peritoneum is so rapid, the paralysis of the intestines so complete, and the distension of the abdominal cavity by the retained gases within the bowels so extensive, that the case is presented almost from the outset as one of acute obstruction of the bowels, in which case proper appreciation of the other conditions is perhaps prevented. For my own part I have been called to more than one case of so-called acute obstruction with enormously distended belly, giving a history of pain indefinitely located, with vomiting early and excessive, shortly becoming stercoraceous, in which the patient's own description of his early symptoms contained no reference to his ileo-cæcal region, and in which the enormous distension prevented recognition of a tumor or any adequate physical examination. And if I have seen such cases, I am sure that many others have, and that it is possible for the most experienced to be deceived. In two such cases it has happened to me to be compelled to operate, rather against my will, simply because the patients desired to take the chances of an operation rather than to die in an otherwise hopeless condition. In both of these

instances a gangrenous appendix, with local gangrene of the cœcum, was found, and the peritoneal cavity contained more or less thin, ichorous discharge, apparently no part of its extent being free from this putrid infection. In at least one of these cases, culture methods gave only pure colon bacillus without mixed forms.

When properly regarded there is nothing stranger about these cases than it is that peritonitis should follow lesions such as internal strangulation of the bowels, strangulated hernia, or other lesions which we have long known to occur. In almost all of these latter instances the peritonitis is of a pure type of colon infection, and yet we all know how frequently fatal it is.

For my own recognition of this course of the disease—that is, for my appreciation of the fact that most cases of so-called spontaneous peritonitis are due to trouble located near the appendix—I have to acknowledge my obligation to Dr. Joseph Price. Since he called my attention to it I have carefully scrutinized my cases and have made bacterial investigations whenever possible, and my convictions have strengthened with the progress of events, until now I have no hesitation in taking the very positive ground which I assume in this paper. Before the French and other investigators had so generally published the fact that the pus from most cases

of so-called perityphlitic abscesses was in fact a pure culture of the colon bacillus. I had established this fact in my own laboratory and from observation in my own practice. I had failed, however, to draw the wider conclusions given above, as most others had failed, until I received the hint from Dr. Price. Since then, with a broader view thus inculcated, I have more carefully investigated my own cases, and my successes or failures, and have frequently asked myself how it happened that others had failed to recognize the same sequence of events; and I have come to the conclusion that for the most part such failure of diagnosis is to be explained on the following grounds:

1. The very common and early diffusion of pain with which many of these cases begin, even those where the phenomena subsequently become strongly localized. This is not always due to ignorance on the part of the patient, for many intelligent people are unable to indicate the place where they first felt the pain of which they so bitterly complain.

2. To the frequent insidiousness of the disease itself, and especially to that with which pus forms in many of these cases.

3. To carelessness in the first examination, and to failure to appreciate the real nature of the lesion in true appendicitis. This is purely a matter of ignorance, and concerns mainly

those men—their number very rapidly diminishing—who diagnosticate all these cases as inflammation of the bowels.

4. To failure to recognize the induration or tumor in the right ileo-cæcal region, which failure may sometimes be well excused because of the distension of the abdomen, the acute sensibility of the part, and the pain which the slightest manipulation produces: in other words, to the impossibility of proper examination.

5. To the fact that some of these cases present, almost from the outset, common signs and symptoms of primary obstruction of the bowels, and that the diagnosis once made by the attendant is not altered to suit the other aspects of the case as they develop. As already indicated, I think we must have charity for those who make this mistake, because to my knowledge it has been made by good and competent general practitioners.

To conclude: The thesis on which I have endeavored to build this paper is as follows:

First. There is no such thing as an idiopathic peritonitis. Every so-called case has a definite origin, which, however, it may not always be possible to easily determine:

Second. Many cases of non-traumatic peritonitis have their origin in the female pelvic organs, and are usually of the type mentioned

under 2 a, *i.e.*, they belong to the staphylococcus and streptococcus forms; but some of them are really cases of colon infection:

Third. Those cases which depend upon perforation after ulceration, escape of gall-stone into the peritoneal cavity, and lesions of this general nature, fall into the septic or putrid forms:

Fourth. Peritonitis due to internal obstruction or strangulated hernia, is usually due to colon infection:

Fifth. Cases of peritonitis which do not originate in the manner already referred to, almost invariably proceed from the appendix vermiformis, and of all these a larger proportion are cases of pure colon infection:

Sixth. The larger proportion of these are fatal unless surgical procedures be used:

Seventh. In every case of peritonitis for which obvious cause is lacking the ileo-cæcal region should be carefully examined, if suspected should be explored, and this exploration may well be made under an anæsthetic with all conveniences at hand for the most formidable kind of operative procedure.

I would like to add in addition to what has been said above, and in a certain way in self-justification, that this paper was prepared early in December before the appearance of at least three different articles which have dealt in a measure with this subject, and by which the statements herein made have been

independently confirmed. These are articles by Dr. M. H. Richardson, of Boston, in the *American Journal of the Medical Sciences*, December, 1893; by George R. Fowler, *Annals of Surgery*, January, 1894; and Dr. Hodenpyl, in the *New York Medical Journal* of Dec. 30th, 1893.

The writer took practically the ground which he has in this paper in another read before the American Surgical Association in June of the past year, and has been practically teaching for two years that the colon bacillus is the source of this infection.

